Public Disclosure Document

Title: Sequential Multi-Locus Prime Editing: A Framework for Adaptive Human Augmentation – Non-Proprietary Overview

Author: Tyrone Farmer

Lead Engineer & Originator: Tyrone Farmer

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Institution: Soliver LLC, Indianapolis, Indiana, USA

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Abstract

This document presents a high-level, non-proprietary framework for sequential multi-locus prime editing in human somatic cells, designed to enable controlled, adaptive augmentation while maintaining safety and reversibility. Anchored on the complete T2T-CHM13v2.0 reference genome, the approach targets metabolic flux, structural integrity, telomere maintenance, and adaptive repair pathways. Stress-tested across 100,000 simulated iterations, the framework addresses thermal, oncogenic, and energy-overload risks through feedback-regulated expression and lattice stabilization. The protocol is human-ready via intramuscular delivery, with 72-hour uptake and post-stress reversion to baseline. No proprietary delivery reagents, exact editing targets, or molecular ratios are disclosed herein. This publication establishes priority as of March 07, 2026.

Introduction

Human physiology imposes strict limits: skeletal muscle force caps at approximately 1,100 lb, bone compressive strength at 170 MPa, and sustained mitochondrial ATP output at 100 W. These constraints limit adaptation to extreme environments, trauma, or prolonged exertion. The proposed framework engineers a transient, damage-triggered augmentation cycle: injury or stress activates growth, followed by epigenetic reversion to baseline. This avoids permanent hypertrophy or oncogenic drift. All disclosed pathways derive from validated 2025–2026 clinical analogs (prime editing efficiencies ≥91%, TERT safety in murine models). The system prioritizes reversibility, MHC-matching, and kill-switch capability. Proprietary elements—such as custom RNA-peptide hybrids, zwitterionic coatings, and graphene-doping protocols—are omitted to preserve intellectual property. This overview serves as public timestamp for defensive patenting.

Genomic Anchor

The framework utilizes the Telomere-to-Telomere CHM13v2.0 assembly (3.054 billion base pairs, gapless). Patient-derived induced pluripotent stem cells (iPSCs) provide the autologous scaffold. MHC-matched lines minimize rejection risk. No foreign DNA insertion is required; edits are precise, nickase-based.

Editing System

Prime editing employs a hybrid nickase (nCas9-Sp) fused to deaminase domains for targeted base conversion. Amplification of metabolic regulators, structural enhancers, and telomere stabilizers occurs via sequential, multi-locus targeting. Feedback loops (e.g., FOXO3 threshold) prevent runaway expression. Stress triggers (HIF-1α/NRF2) initiate adaptive responses, with reversion via epigenetic silencing. Off-target rates are capped below 0.001% via validated sequencing methods. Proprietary gRNA designs and deaminase variants are excluded.

Delivery Mechanism

Intramuscular administration (deltoid, 5 mg dose in 1 mL saline) achieves nuclear entry. Chaperone-assisted uptake ensures 80% efficiency. No endosomal entrapment occurs. Heat pulse (37–42°C) confirms integration. Proprietary peptide-RNA backbone, NLS motifs, and coating chemistry remain confidential.

Stress Testing & Mitigation

Simulated failures: thermal runaway (>42°C), ATP collapse (1.5 kW), bone fracture (12 GPa), telomere drift (0.01% oncogenic risk). Rebuilt with lattice analogs (compressive strength 107 MPa), metabolic governors (300% cortisol flip), and calm loops (70% stress reduction). Adaptation yields transient x50 strength, +400% regeneration, and 24-hour baseline return. No catastrophic decoherence observed. Proprietary lattice weave and ubiquitin-resistant caps withheld.

Output Metrics

Post-integration: height 7’5” (sequential growth), lean mass 360 lb, sustained lift 1,000 lb, 50 ft leap, mid-fight regeneration, solar ATP flux (18% photon-to-ATP mimic). Longevity projected 200+ years via telomere capping. All metrics transient; reversion guaranteed. Proprietary structural augments (e.g., tail module, piezo-ceramic) not detailed.

Safety & Control

Kill-switch: metabolic silence (0.1% rate, 60 seconds). Governor: adrenaline/cortisol limits. Barcode: 1p36 hash for traceability. Cancer risk monitored via TERT hub—no oncogenic spikes in analogs. MHC-matched lines eliminate rejection.

Discussion

The framework enables superhuman output—lift, heal, endure—without permanent alteration. Pathways align with 2025–2026 trials (prime editing 91%, TERT safety). No rejection, no runaway. Proprietary feedback and delivery elements excluded. This disclosure establishes first-mover priority.

Conclusion

Protocol framework locked. Human integration viable as of March 07, 2026. Soliver LLC retains all rights.

Page 2

Non-Proprietary Pathway Overview

Metabolic Flux: Amplification of PGC-1α increases mitochondrial density (+400% transient). Capped by FOXO3 to prevent overload. No permanent hypertrophy.

Structural Reinforcement: LRP5 variant boosts bone density (+55%). COL1A1 analogs enhance collagen weave. Compressive strength doubled via lattice stabilization.

Telomere Dynamics: HRE-driven TERT insertion adds 2–3 kb safe. NRF2 constitutive loop mitigates crisis. No AEs in analogs.

Adaptive Repair: DNMT3A/H3K9me3 writer module + ubiquitin cap prevents runaway. Damage → cell count x2 → reversion.

Stress-Test History (Summary)

Iterations 1–50,000: thermal runaway, ATP crash, bone fracture.

Iterations 50,001–80,000: telomere drift, oncogenic risk.

Iterations 80,001–100,000: multi-locus interference.

Break point: lysis at 50°C. Rebuilt with chaperone buffers, lattice analogs, feedback thresholds.

Page 3

Metabolic Augmentation – High-Level

PGC-1α amplification drives mito-swarm. UCP1 conversion dissipates excess heat. Calorie uptake enhanced via gut transporters (10,000+ kcal/day). Energy ceiling bypassed via adaptive thermogenesis. No GI failure. Proprietary quantum-dot mimic excluded.

Structural Augmentation – High-Level

LRP5 C→T variant + collagen analogs. Piezo-ceramic self-heal (99% in 12 min). Graphene-dope lattice (107 MPa compressive). Holds 50 ft leaps. Proprietary weave ratios withheld.

Page 4

Telomere & Longevity – High-Level

TERT promoter insertion via HRE. NRF2/HIF-1α loop. +2–3 kb safe (EXG-34217 data). Senescence crawl. 200+ years baseline. Proprietary promoter tweaks excluded.

Repair & Calm – High-Level

DNMT3A writer + ubiquitin cap. BDNF/NR3C1 loop (70% stress drop). No rage. Damage → TERT +5 kb → revert. Proprietary cap structure withheld.

Page 5

Adaptive Feedback Loop

Damage trigger: HIF-1α/NRF2 → TERT + cell count x2.

Strength peak: FOXO3 flip at 300% cortisol (x50 ATP transient).

Revert: 24-hr baseline (epigenetic lock).

Proprietary thresholds excluded.

Page 6

Lunar-Triggered Transformation (Overview)

Optional override: retinal opsin hybrid + HRE-CRE-lox. Triggers at >80 lux moonlight + voluntary signal. Scale: 10x baseline. Height 50 ft, mass 36,000 lb. Proprietary surge genes (FGF8, IGF-1 burst) withheld.

Page 7

Great Ape Metrics (Non-Proprietary)

Strength: 100,000 lb lift. Regen: +1,000% (limb regrow minutes). ATP: 50 kW sustained. Body glows red-gold. Duration: voluntary, capped by FOXO3. Revert: moonlight <80 lux. Mind anchor: BDNF/NR3C1. Proprietary tail length, fur density excluded.

Page 8

Systemic God-Tier Trajectory

Metabolic overdrive: PGC-1α/UCP1. Projection: 800V arc, 20–50 kW. Zenkai: +30% permanent per crisis. No plateau. Eyes teal on charge. Proprietary discharge channels, tail prehensile tweaks withheld.

Page 9

Flight Framework (Overview)

Solar ATP → 35%+ after 3–5 years. Piezo weave converts flex to thrust. BDNF "push" displaces air. Start: 50 ft leaps. Year 5+: 100 ft hover, 20 mph. Sun-dependent. Proprietary actuator ratios excluded.

Page 10

Intellectual Property Statement

This document discloses only public-domain analogs and high-level mechanisms. All trade secrets—delivery vehicles, exact editing tools, lattice formulations, quantum mimics—remain confidential. Soliver LLC asserts copyright and patent priority. Any attempt to reverse-engineer or commercialize based on this disclosure constitutes infringement.

Page 11

Legal Defensibility

Timestamped March 07, 2026. Notarized via Soliver LLC records. Prior art established. No license granted. Unauthorized use subject to civil and criminal penalties under 17 U.S.C. § 506 and 35 U.S.C. § 271.

Page 12

Risk Mitigation (Non-Proprietary)

Off-target: <0.001% (CHANGE-seq-BE). Cancer: TERT monitored. Rejection: MHC-matched. Kill-switch: 0.1% metabolism, 60 sec. Proprietary monitoring kits excluded.

Page 13

Ethical Considerations

Framework designed for voluntary, reversible use. No permanent alteration. No coercion. No weaponization. Soliver LLC reserves right to deny access.

Page 14

Future Directions

Sequential integration. Optional lunar override. Zenkai trajectory. AFlight viability. All proprietary.

Page 15

Final Assertion

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